Acute Peat Smoke Inhalation Increases Blood Pressure and Cardiac Arrhythmia Risk in Rats

AK Farraj[1], BL Martin[2], LC. Thompson[1], Y Kim[2], C King[1], S Snow[1], M Schladweiler[1], N Haykal-Coates[1], I George[3], M Higuchi[1], MI Gilmour[1], UP Kodavanti[1], MS. Hazari[1]

[1]Environmental Public Health Division, US EPA, RTP, NC, [2]Oak Ridge Institute for Science and Education, Oak Ridge, TN, [3]NRMRL, US EPA, RTP, NC

Wildland fires are increasingly linked to adverse health impacts related to poor air quality. For instance, short-term exposure to smoldering peat (SP) air pollution in eastern North Carolina during separate burns in 2008 and 2011 led to increased cardiopulmonary emergency room visits. Little is known, however, about potential biological mechanisms and the influence of exposure levels. The purpose of this study was to assess the cardiovascular impacts of acute exposure to SP biomass smoke in rats and relate them to potential autonomic and inflammatory mechanisms as well as levels of volatile organics, gases and particulate matter (PM). Three month-old male Wistar-Kyoto rats were exposed once, for 1-hr, to filtered air (FA) or SP smoke, generated using an automated control tube furnace system wherein PM was diluted to low (LP; 0.36 mg/m3) or high (HP; 3.76 mg/m3) concentrations. Rats were monitored for heart rate (HR), blood pressure (BP), electrocardiogram (ECG), and heart rate variability (HRV), an indicator of autonomic tone, before, during and after exposure. Systemic markers of inflammation and sensitivity to aconitine-induced cardiac arrhythmia, a measure of latent myocardial vulnerability, were assessed in separate cohorts of rats 24-hr after exposure. PM size (LP = 0.4 - 0.5 microns vs. HP = 0.8 - 1.2 microns) and proportion of organic carbon (LP = 77% vs. HP = 65%) varied with exposure level. Exposure to HP, but not LP, caused increases in systolic (11.6%; p=0.0503) and diastolic BP (15.6%; p=0.02) and a decrease in HR (p=0.0654) relative to exposure to FA (ECG and HRV data are forthcoming). By contrast, only exposure to LP increased sensitivity to aconitine-induced cardiac arrhythmia relative to exposure to FA ($p<0.01$). Few changes in systemic markers were evident. Taken together, HP caused overt responses not present with LP that were potentially mediated by autonomic responses. Perhaps of greater concern is the finding that exposure to lower levels that better approximate most human ambient exposures to smoke plumes only caused latent effects, indicating that the effects of exposure may be insidious. This abstract does not reflect US EPA policy.